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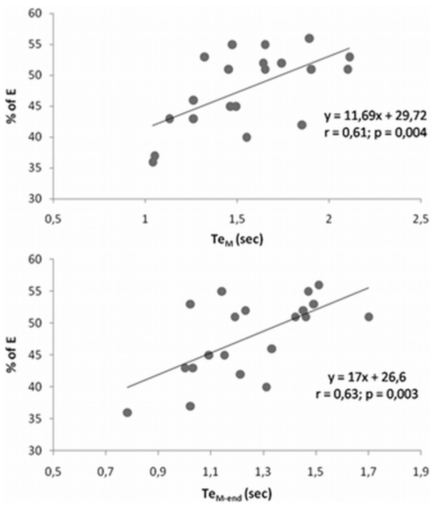
Exercise respiratory cycle time components in patients with emphysema

Alessandro Maria Ferrazza, Patrizia Paoletti, Gabriele Valli, Paolo Onorati, Dario Martolini, Michela Mordenti, Francesco Vaccaro, Paolo Palange.
Department of Public Health and Infectious Diseases, Lung Function Unit, Sapienza University of Rome, Italy

Background: We have recently demonstrated that in patients with COPD the severity of emphysema (E) measured by high resolution computed tomography (HRCT) correlated with: ratio VTpeak/FEV1; VE/VCO2 slope and PETCO2 values at peak exercise. The aim of this study was to further investigate if exercise respiratory cycle time components correlated with % of E measured by HRCT.
Method: Twelve patients (age = 65±8 yrs; FEV1 = 55±17%pred) with moderate to severe E (quantified by lung HRCT as % voxels < −910 HU) were evaluated with incremental cardiopulmonary exercise testing (CPET). Mean inspiratory time (TiM), mean total respiratory cycle time (TtotM), mean expiratory time during exercise (TeM) and mean expiratory time during the last third of exercise (TeM-end), has been calculated.
Results: Both TeM and TeM-end had a good linear correlation with % of E (r = 0,61; p = 0,004 and r = 0,63; p = 0,003).



Moreover, by dividing the patients in two groups based on the % of E (>50% and <50%), we observed that patients with higher % of E had longer TeM (TeM: 1,72±0,26sec vs 1,34±0,27sec, p = 0,005) and TeM-end. A good linear correlation has been observed also between TeM and PETCO2 and VE/VCO2 (r = 0,64; p = 0,002 and r = 0,7; p = 0,0005). TeM did not correlated with resting lung function values or inspiratory capacity (IC).
Conclusion: The data confirm that distinct physiologic response pattern can be detected at CPET in these patients.

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Skeletal muscle oxygenation during exercise in patients with chronic respiratory failure

Kazuyuki Tabira[1], Jun Horie[2], Hiromasa Fujii[3], Toshio Aida[3]. [1]*Department of Physical Therapy, Faculty of Health Science, Kio University, Kitakatsuragi-gun, Nara, Japan;* [2]*Department of Rehabilitation Science, Kobe International University, Kobe, Hyogo, Japan;* [3]*Department of Respiratory Medicine, Osaka Prefectural Medical Center of Respiratory and Allergic Disease, Osaka, Japan*

Background: Muscle oxygenation correlates with systemic oxygen uptake (VO_2) in normal subjects, however whether this relationship exists chronic respiratory failure (CRF) patients remains unclear.
Objectives: The purpose of this study was to investigate the influence of skeletal muscle oxygenation on VO_2 during exercise in CRF patients.
Methods: Ten chronic obstructive pulmonary disease and two sequelae of pulmonary tuberculosis patients performed an incremental cycle ergometer exercise test. We measured ventilation, pulmonary gas exchange and SaO_2. We also measured tissue oxygen saturation (StO_2) in the vastus lateralis with continuous-wave near-infrared spectroscopy. We calculated the muscle oxygen extraction rate (MOER) based on SaO_2 and StO_2. In addition, we performed regression analysis to examine the relationships between the VO_2 obtained during exercise testing and the mean values of SaO_2, StO_2, heart rate (HR), and MOER for each 30-second interval of the tests. Finally, we analysed the relationships between the peak value of oxygen uptake (VO_{2peak}) and the slopes of HR/VO_2, SaO_2/VO_2, StO2/VO_2, and MOER/VO_2.
Results: With the increasing exercise intensity, many subjects showed a gradual decrease in StO_2 and SaO_2, but a gradual increase in HR and MOER. VO_2 was negatively correlated with StO_2 and SaO_2, and was positively correlated with HR and MOER. However, VO_{2peak} was not correlated with any of the slopes.
Conclusions: VO_2 is highly influenced by oxygen utilization in exercising muscles, as well as by blood oxygenation levels and cardiac function. However, the impact of skeletal muscle utilization during exercise on VO_{2peak} varied greatly among the patients.

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Inspiratory muscle constraint during exercise in patients with pulmonary arterial hypertension

Barbara Joureau-Harasse[1], Gilles Garcia[2,3,4], Christian Straus[1], Elise Arnaud Macari[2,3,5], Xavier Jaïs[2,3,5], David Montani[2,3,5], Laurent Savale[2,3,5], Olivier Sitbon[2,3,5], Gérald Simonneau[2,3,5], Marc Humbert[2,3,5], Thomas Similowski[1,6], Pierantonio Laveneziana[1,2,3,6]. [1]*Equipe de Recherche ER 10 UPMC, Laboratoire de Physio-Pathologie Respiratoire, Faculté de Médecine Pierre et Marie Curie (site Pitié-Salpêtrière), Université Paris 6, France;* [2]*Faculté de Médecine, Université Paris-Sud, Le Kremlin-Bicêtre, France;* [3]*Centre Chirurgical Marie Lannelongue, INSERM U999, Le Plessis-Robinson, France;* [4]*Service de Physiologie, Assistance Publique Hôpitaux de Paris, Hôpital Antoine Béclère, Clamart, France;* [5]*Service de Pneumologie et Réanimation Respiratoire, Centre National de Référence de l'Hypertension Pulmonaire Sévère, Assistance Publique Hôpitaux de Paris, Hôpital Antoine Bécl[egrave;* [6]*Service de Pneumologie et Réanimation Médicale, Assistance Publique-Hôpitaux de Paris, Groupe Hospitalier Pitié-Salpêtrière, Paris, France*

We examined the impact of potential inspiratory muscle constraint on dynamic operating lung volumes response during symptom-limited incremental cardiopulmonary cycle exercise testing (CPET) in patients with pulmonary arterial hypertension (PAH).
Thirty-three young non-smoking PAH patients (idiopathic=26; heritable=7) with normal body mass index and no spirometric evidence of obstructive ventilatory defect (FEV_1/FVC=115±10% predicted) performed a CPET to limit of tolerance. Ventilatory profile, operating lung volumes [derived from inspiratory capacity (IC) measurements] and inspiratory flow reserve (IFR), an indirect index of inspiratory muscle constraint/fatigue,were assessed throughout CPET.
Twenty-two patients (67%) decreased IC (i.e., dynamic hyperinflation) throughout exercise by 0.50L (PAH-H), whereas the remaining patients (33%) increased IC by 0.36L (PAH-NH). V'E and V'O2 at peak exercise were comparable between the two groups. Despite these differences in operating lung volumes response, IFR at peak exercise was not statistically different between PAH-H and PAH-NH (1.9±1.0 vs 2.0±0.8L/s, p=0.7).
Both PAH-H and PAH-NH achieved inspiratory tidal flows that approached a similar percentage of the maximal available inspiratory flows (i.e., similar IFR), suggesting that the inspiratory flow-generating reserve of the inspiratory muscles at peak exercise was similar (but occurred at different operating lung volumes). The presence of inspiratory muscle constraint/fatigue and its contribution in modulating the dynamic operating lung volumes response to CPET is unlikely.
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A metabolic evaluation of a group of obese children: Oxygen consumption (VO2) and power

Martina Tubaro[1], Ilenia Benfatto[1], Mario Canciani[1], Francesca Valent[2].
[1]*Allergology and Pulmonology Unit, Department of Paediatrics, DPMSC, University of Udine, Italy;* [2]*Hygiene Department, School of Medicine, DPMSC, University of Udine, Italy*

Obesity, which has been considerably increasing in the last decades, is related to significant metabolic alterations.
The aim of this study was to evaluate whether this condition can also alter the VO2 (considered as the best assessment of cardiorespiratory form in healthy children) and the power in a group of obese children compared to a group of control children.
We tested 98 children: 42 controls and 56 obese children. Each child underwent the incremental test on the cycle ergometer according to a protocol of increasing effort.
VO2 values were 2027.6 ml/min for the group of controls and 2078.0 ml/min for the obese children ($p>0.05$); instead, the values of VO2/kg were lower in the obese children 32.3 ml/kg compared to the group of controls 38.7 ml/kg/min ($p<0.01$). The higher value of power was obtained in the group of control children (140 watt) and lower in the obese (120 watt) ($p<0.05$); this parameter normalized for the mass was higher in the group of controls (2.7 watt/kg) compared to obese children (1.9 watt/kg) ($p<0.01$).
Given the greater body mass of obese due to an increased fat component, it is not surprising that the VO2 is higher. These subjects must in fact move a large mass, with a considerable consumption of energy, to the detriment of their ability to work. The VO2/kg values are lower in the obese as cardiac or metabolic disease limit the peripheral oxygen consumption. In obese children the power values are lower as the lean body mass consumes less oxygen that fat body mass, it produced less work therefore less power. Power/kg is lower in obese children due to their larger mass.